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Case Report

A RARE PRESENTATION OF CANDIDIASIS: CONGENITAL CUTANEOUS CANDIDIASIS IN A NEONATE BORN TO AN ASYMPTOMATIC MOTHER

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ABSTRACT
Neonatal candidiasis can either be acquired at birth by ascending infection through birth canal, or in postnatal period via direct contact. Congenital cutaneous candidiasis (CCC) is a rare disorder that presents within the first six days of life in neonates exposed in utero. It is usually seen in infants with maternal candidal vulvovaginitis. We report a case of a full term neonate, presenting with generalized maculo papular skin eruptions three days after birth, born to an asymptomatic mother. The manifestation of CCC ranges from diffuse skin eruptions without any systemic symptoms to severe systemic manifestations. Clinical features, direct microscopic examination of specimen, and appropriate cultures are useful in differentiating the lesions from other more common dermatoses of the neonatal period. Although, this disorder is usually benign and self-limiting in full term infants with normal birth weight, it can be life-threatening in premature and low birth weight neonates.

Keywords: *Congenital Cutaneous Candidiasis, Neonate*

INTRODUCTION
Candida species are part of the lower genital tract flora in 20-50 % of healthy asymptomatic women especially during pregnancy (Pradeep *et al.,* 1998). Neonatal candidiasis can either be acquired at birth, by ascending infection through birth canal, or in postnatal period via direct contact with nursing staff (Lane, 1995). Neonates exposed to *Candida* species in-utero can present with Congenital Cutaneous Candidiasis (CCC), a rare neonatal fungal infection, within six days of birth. It is usually seen in infants with maternal vulvovaginal candidiasis (Darmstadt *et al.,* 2000). Less than 100 cases of CCC have been reported worldwide in the literature and a few from India (Torres-Alvarez *et al.,* 2013**;** Srinivas and Bhardwaj, 2014**)**. We hereby report a case of CCC in a full term neonate born to an asymptomatic mother without any other associated risk factors.

CASES
A full term female baby weighing 2.75 kg was born to a 30-year old third gravida mother by elective lower segment caesarean section (LSCS) with an indication of previous caesarean. Mother did not have any history of vaginal discharge or itching during pregnancy. Prior to onset of labor there was no per vaginal bleed, or premature rupture of membrane (PROM). There was no history of intrauterine devices being used in the past and per vaginal examination confirmed the same. Mother was seronegative for Venereal Disease Research Laboratory (VDRL) test and Human Immunodeficiency Virus (HIV), there were no rashes on her body neither she had any history of contact with chicken pox during perinatal peroid. At birth baby had mild respiratory distress and kept in neonatal intensive care unit for monitoring. Baby improved within few hours and a diagnosis of transient tachypnea was made. She was accepting breast feed & was not on any medication. On 3rd day of life baby developed generalized, erythematous macular eruptions over body especially on trunk, back and extremities, sparing face, palms and soles, few lesions had fine scaling (figure 1). The eruption soon became confluent at some sites and progressed to vesicles and pustules; no other systemic manifestations were observed. Skin scraping and vesicle fluid

were collected and subjected to direct (KOH) mount microscopic examination and Gram's stain. Budding yeast cells with pseudohyphae were demonstrated. Sample was also inoculated into Blood agar (BA) and Saboraud's dextrose agar (SDA) with antibiotics and incubated at 37º C and 25ºC, respectively. After 24 hrs of incubation white creamy pasty colonies were observed on BA and SDA. The isolate was identified as *Candida albicans* on the basis of gram's staining, positive germ tube test, demonstration of chlamydospores on corn meal agar and sugar assimilation and fermentation tests. Blood and urine culture for bacteria and fungi obtained on admission to intensive care unit were negative. Total leucocyte counts and differential leucocyte counts were within normal limits. Mother was tested seronegative for Toxoplasma, Rubella, Cytomegalovirus, Herpes simplex (TORCH), test. A high vaginal swab collected from mother demonstrated the presence of *C. albicans* on culture. The diagnosis of CCC was made on the basis of clinical picture, positive KOH mount and culture of the skin lesions. Clotrimazole (1%) cream was applied on the lesions which resulted in a dramatic improvement and lesions healed with superficial desquamation of the involved skin after approximately four days, local ointment was discontinued and mother and child were discharged from the hospital.

DISCUSSION

Congenital candidiasis was first reported in 1960 (Sonnenschein *et al.,* 1964). Prematurity and the presence of an intrauterine foreign body (for e.g intrauterine device, cervical sutures, tampons), PROM are reported to be associated with this condition, however cases have also been reported in healthy full term neonates, in the absence of associated risk factors (Pradeep *et al.,* 1998; Darmstadt *et al.,* 2000; Torres-Alvarez *et al.,* 2013).

Exact pathogenesis of CCC is not known but many cases CCC have occurred in neonates born with clinically intact chorioamniotic membranes as observed in our case (Darmstadt *et al.,* 2000) .There is evidence that *C. albicans* can penetrate intact membranes. Once the membranes are penetrated, organisms are hypothesized to spread from the amniotic fluid to the skin and into the pulmonary and gastrointestinal tract, in case the fluid is aspirated or swallowed (Darmstadt *et al.,* 2000; Jagtap *et al.,* 2011). Acid proteinase secreted by C. albicans has keratolytic activity and is thought to facilitate initiation of cutaneous candidiasis and assist in invasion (Ray and Payne, 1990). Demostration of pseudohyphae in direct skin sample also indicates towards the pathogenic potential of the *C. albicans* strain rather than a mere colonizer.

CCC can produce a spectrum of disease ranging from a diffuse skin eruption in the absence of systemic illness to severe systemic disease resulting in fetal demise or early neonatal death. However the most common presentation is an acute generalized eruption of erythematous macules, papules and or pustules with a benign outcome (Darmstadt *et al.,* 2000). The risk of dissemination is increased if there is an evidence of respiratory distress or other signs of sepsis in immediate neonatal period, low birth weight (<1500 gm), administration of broad spectrum antibiotics, extensive instrumentation during delivery and a positive blood, urine, or Cerebrospinal fluid culture (Johnson *et al.,* 1981).

The differential diagnosis of macupapular rashes in neonates are CCC, staphylococcal scalded skin syndrome, toxic shock syndrome, erythema toxicum, transient neonatal postular melanosis, drug eruptions, herpes simplex, infantile seborrhoeic dermatitis, atopic dermatitis, psoriasis, congenital syphilis, *Listeria monocytogenes* and *varicella zoster* virus infections (Table 1) and (Table 2) (Darmstadt *et al.,* 2000; Jagtap *et al.,* 2011). The vague clinical picture of CCC resembling other skin eruptive conditions, may lead to misdiagnosis of such cases. Although, this disorder is usually benign and self-limiting in full term infants with normal birth weight, it can be life-threatening in premature and low birth weight neonates (Johnson *et al.,* 1981). So a high index of suspicion should be maintained by clinicians and clinical microbiologist, to prevent a benign treatable condition from taking a severe and protracted course, and avoid misdiagnosis of such cases.

Therefore, we highlight the importance of direct microscopic examination of a KOH wet mount and appropriate bacterial/fungal culture, of such skin lesions, to differentiate CCC from other causes of maculopapular eruptions, in neonates born to asymptomatic mothers.

Table 1: Differential diagnosis of Congenital Cutaneous Candidiasis according to their clinical manifestations

Diseases	Clinical Manifestations
Staphylococcal scalded skin syndrome	Skin tenderness, superficial blisters/bullas, positive Nikolsky sign.
Toxic shock syndrome	Skin tenderness, hypotension/shock
Erythema toxicum	Erythematous macules with overlying white or yellow papules or pustule, which resolves within 2 weeks, frequently individual lesions appear and disappear within minutes.
Transient neonatal pustular melanosis	Vesicles, superficial pustules, and pigmented macules are present at birth. They occur on the chin, neck, forehead, chest, buttocks, back, and, less often, on the palms and soles. The vesicles and pustules rupture easily and resolve within 48 hours. The brown macules may persist for several months.
Drug eruptions (ceftriaxone and vancomycin)	Generalised skin rashes.
Herpes simplex	Watery blisters on skin , oral mucosa and genitalia.Lesions heal with a scab formation.
Infantile seborrhoeic dermatitis	Cradle cap, accentuation in the skin folds of the neck, axillae, and nappy area
Atopic dermatitis	Encrusted eczema on the scalp and face, generalised eczematous skin
Psoriasis	Erythematosquamous patches, can be pustular
Congenital syphilis	Maculopapular (coppery-brown) skin rash followed by desquamation, blistering and crusting, prominent on the palms and soles. Delays in development, seizures, fever, hepatosplenomegaly, anemia, jaundice and snuffles.
Listeria monocygenes infection	Respiratory difficulty like cyanotic episodes, rapid breathing, and grunting.
Congenital Varicella	Skin lesions in dermatomal distribution, neurologic defects, eye diseases and skeletal anomalies

Table 2: Diagnostic modalities used for differentiation of congenital cutaneous candidiasis from other causes of erythematous macules

Diseases	Contributory evidence	Microbiological evidence
Staphylococcal scalded skin syndrome	History of preceding infection by *Staphylococcus aureus*	Demonstration of toxin producing strain of *Staphylococcus aureus* from skin lesions.
Toxic shock syndrome	Concomitant maternal infection	From skin swab demonstration of toxin producing strains of *Staphylococcus aureus* or *Streptococcus pyogenes*.
Erythema toxicum	Fluid from lesions shows many eosinophils; High levels of circulating eosinophils	Nil
Transient neonatal pustular melanosis	Nil	Tzanck smear/gram's stain from pustule reveals predominance of neutrophils, occasional eosinophils.
Drug eruptions (ceftriaxone and vancomycin)	History of administration of antibiotics	Nil
Herpes simplex	Nil	Positive direct fluorescent antibody test from skin scraping and fluid, Demonstration of multinucleated giant cells in Tzanck smears of skin lesions.
Infantile seborrhoeic dermatitis	Diagnosis mailnly clinical	Nil
Atopic dermatitis	Family history of atopy, Raised IgE and eosinophilia	Nil
Psoriasis	Positive family history, Skin biopsy shows hyper and parakeratosis, microabscesses	Nil
Congenital syphilis	Nil	Skin lesions or nasal discharge (snuffles) examined for spirochetes by darkfield microscopy or by direct fluorescent antibody techniques.
Listeria monocygenes infection	Meconium-stained amniotic fluid	Isolation of bacteria from skin lesions, blood or cerebrospinal fluid culture. •
Congenital Varicella	Maternal varicella infection	*Varicella zoster* virus specific IgM detection, Molecular diagnosis and in situ hybridization.

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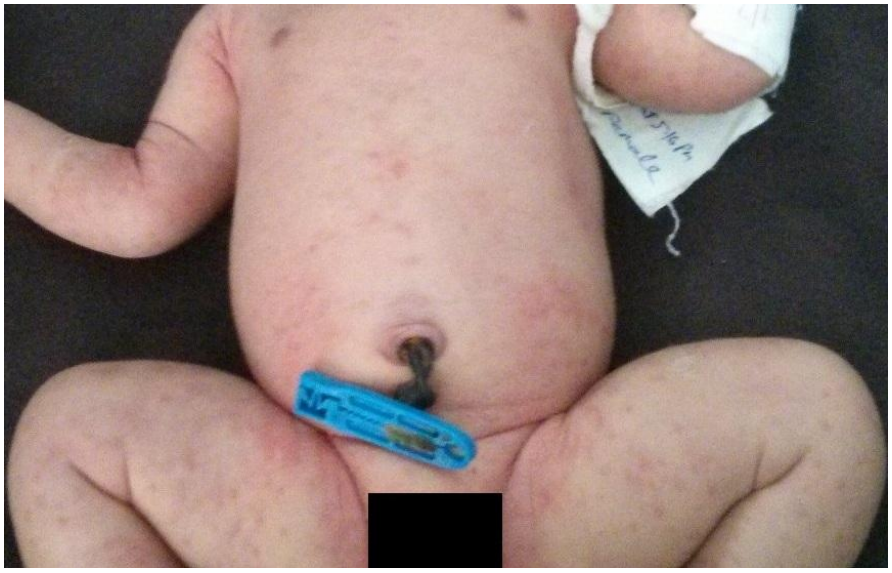

Figure 1: Full-term neonate with congenital cutaneous candidiasis, presenting with generalized erythematous macules

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